The contribution of the external carotid artery to cerebral perfusion in carotid disease

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Purpose: In the presence of carotid occlusion, the external carotid artery (ECA) becomes an important source of cerebral blood flow, especially if the circle of Willis is incomplete. The contribution of the ECA to hemispheric blood flow in patients with severe ipsilateral carotid stenosis has never been previously investigated.
Methods: One hundred eight patients were monitored during sequential cross-clamping of the external (ECA) and then ipsilateral internal carotid artery (ICA) during carotid endarterectomy using transcranial Doppler sonography (TCD) (Neuroguard CDS, Los Angeles, Calif), to measure middle cerebral artery blood flow velocity, and near-infrared spectroscopy, to measure regional cerebral oxygen saturation (CsO_2) (Invos 3100A; Somanetics, Troy, Mich).
Results: On the ipsilateral ECA cross-clamp, the median fall in CsO_2 was 3% (interquartile range, 1%-4%; P <.0001). On addition of the ICA cross-clamp there was a further fall of 3% and a total fall of 6% (3%-9%; P <.0001). The median percentage fall in middle cerebral artery blood flow velocity on ECA clamping was 12% (4%-24%; P <.0001); on ICA clamping it was 48% (25%-74%; P <.0001). Falls in TCD on ECA clamping were greater with increasing severity of ipsilateral ICA stenosis. The correlation between CsO_2 and TCD on external clamping, although less strong than that on internal clamping, was statistically significant r = 0.32; P = .01; Spearman rank correlation).
Conclusions: The falls in TCD and CsO_2 were of a similar order of magnitude and must therefore reflect a fall in cerebral perfusion. The ipsilateral ECA contributes significantly to intracranial blood flow and oxygen saturation in severe carotid stenosis. (J Vasc Surg 2000;31:989-93.)

The importance of collateral circulations to the brain are well known and include large interarterial connections through the circle of Willis, small interarterial connections arising from leptomeningeal collaterals on the cerebral surface, and extracranial-to-intracranial connections.[1-3] The most important of these extracranial-to-intracranial collaterals is through the external carotid artery (ECA), collateralizing primarily through the periorbital plexus. In the presence of internal carotid artery (ICA) occlusion, the ipsilateral ECA is likely to be essential in patients where the circle of Willis is incomplete. The importance of the ECA collateral circulation in patients with severe carotid disease has never been adequately investigated. We therefore studied the contribution of the ipsilateral ECA to cerebral perfusion in symptomatic patients undergoing carotid endarterectomy for >70% stenosis of the ICA.

METHODS

One hundred eight patients gave informed consent to be studied during carotid endarterectomy for symptomatic stenosis of the ICA of 70% or more. Preoperative symptoms included transient ischemic attacks in the relevant hemisphere or ipsilateral amaurosis fugax. Preoperative ICA stenosis was measured noninvasively using duplex Doppler ultrasound scanning rather than angiography to avoid the risk of stroke. Intracranial disease was not assessed.

Competition of interest: nil.
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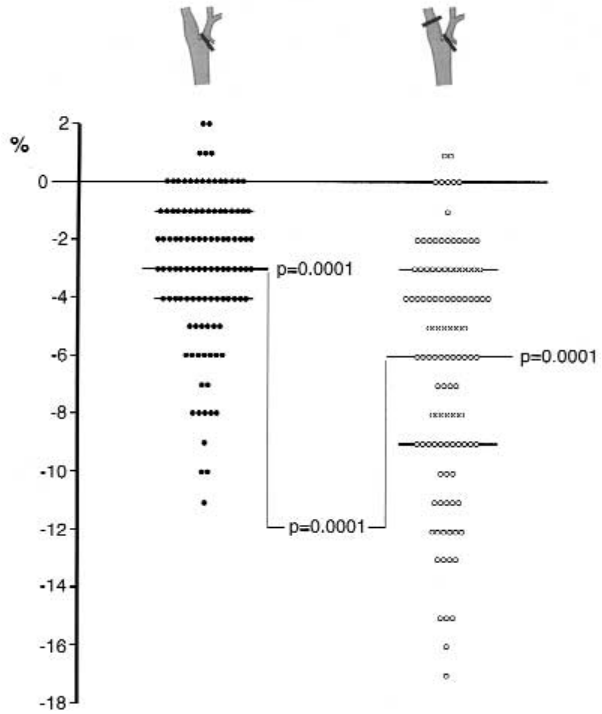

Fig 1. Absolute fall in the percentage CsO_2 on clamping the ECA and then on additional clamping of the ICA for each patient. Median and interquartile ranges are shown, and statistical comparison is by the nonparametric Mann-Whitney *U* test. *CsO_2*, Cerebral oxygen saturation; *ECA*, external carotid artery; *ICA*, internal carotid artery.

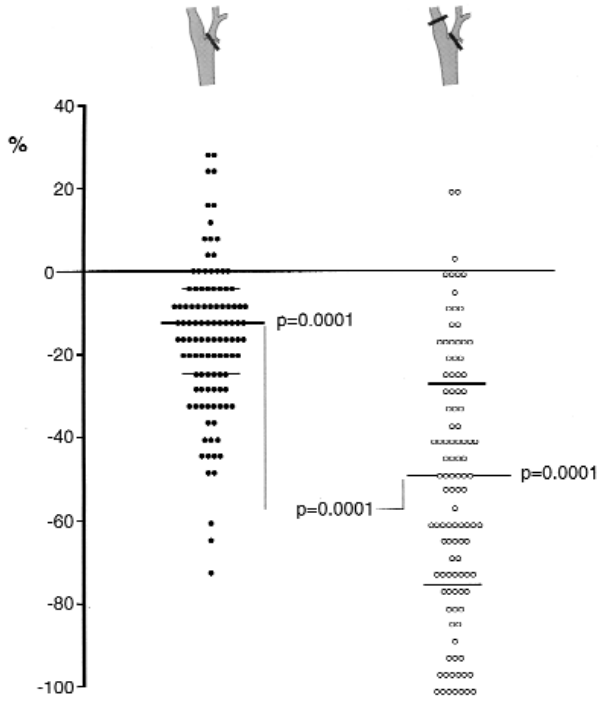

Fig 2. Percentage fall in middle cerebral artery blood flow velocity by TCD on clamping the ECA and then on additional clamping of the ICA for each patient. Results are expressed as median and interquartile ranges and demonstrate significant falls in middle cerebral blood flow on both ECA and ICA cross-clamp (Mann-Whitney *U* test). *ECA*, External carotid artery; *ICA*, internal carotid artery; *TCD*, transcranial Doppler sonography.

Patients were monitored using transcranial Doppler (TCD) (Neuroguard CDS, Los Angeles, Calif) placed over the temporal window, to measure middle cerebral artery blood flow velocity, and near-infrared spectroscopy, to measure regional cerebral oxygen saturation (CsO_2) (Invos 3100A; Somanetics, Troy, Mich). The oximetry sensor was placed over the middle cerebral artery territory, not on the forehead, as the manufacturers recommend. We have shown previously that this is more sensitive to hypoperfusion in the middle cerebral artery territory and improves correspondence between middle cerebral flow and oxygen saturation during carotid clamping and declamping.[4,5] This usually entailed shaving a small patch of hair on the parietal scalp to place the sensor just above the temporalis muscle.

General anesthesia was standardized, with end-tidal carbon dioxide levels kept within normal limits to preserve autoregulation. Blood pressure was maintained within 40 mm Hg of preoperative systolic levels using standard vasoactive drugs. Ipsilateral mean middle cerebral artery blood flow velocity was measured with TCD, and regional CsO_2 was measured with reflected near-infrared spectroscopy. The following steady states were recorded: postinduction before surgery, 3 minutes after clamping the ECA alone, and 3 minutes after additional clamping of the ICA. Three minutes were chosen so as not to delay shunt insertion where indicated and because our experience suggests that both TCD and CsO_2 stabilize within this period after ICA clamping. In practice both TCD and CsO_2 tend to recover over the ensuing 5 minutes as collateral circulations open up.

RESULTS

The mean age was 68 years (range, 51-86 years), and 72% of the patients were men. All patients had >70% ICA stenosis, and there was <70% ipsilateral ECA stenosis in 78% of the patients. There was >70% contralateral ICA stenosis in 42% of the patients. The median absolute fall in CsO_2 was 3%

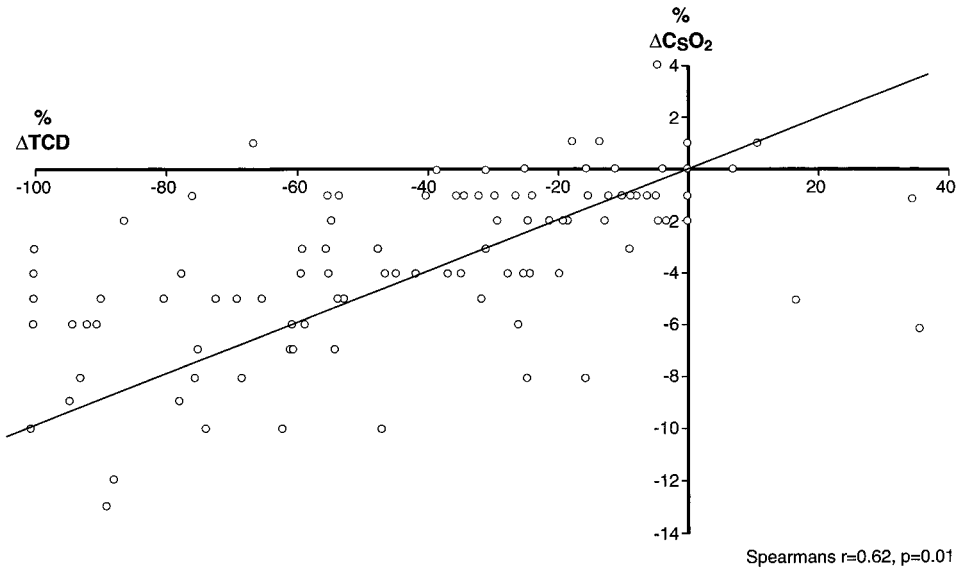

Fig 3. Correlation between the fall in CsO_2 and fall in middle cerebral blood flow velocity by TCD on ICA clamping (Spearman rank correlation coefficient, $r = 0.62$; $P < .01$). *CsO_2*, Cerebral oxygen saturation; *ICA*, internal carotid artery; *TCD*, transcranial Doppler sonography.

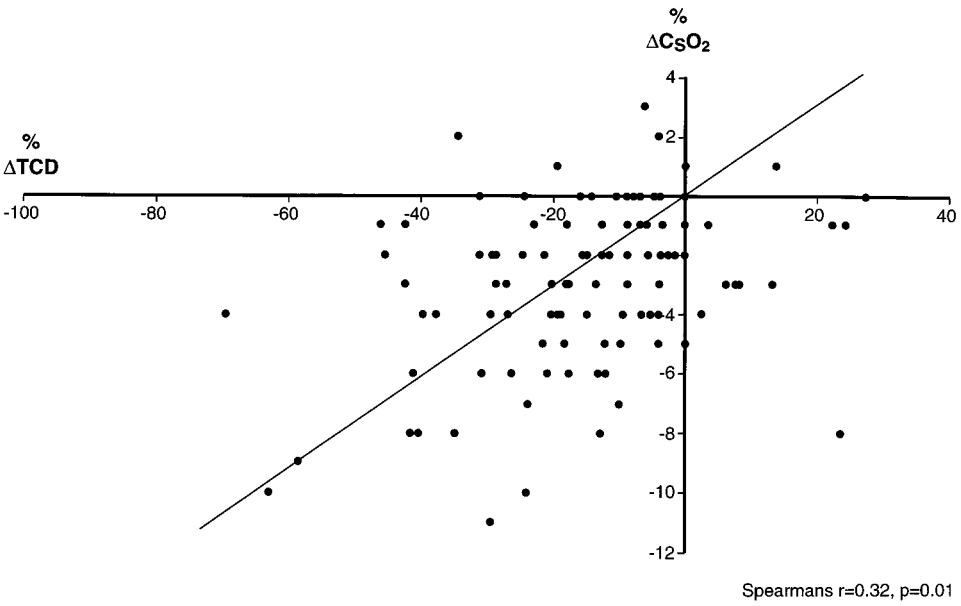

Fig 4. Correlation between the fall in CsO_2 and fall in middle cerebral blood flow velocity by TCD on ECA clamping (Spearman rank correlation coefficient, $r = 0.32$; $P < .01$). *CsO_2*, Cerebral oxygen saturation; *ECA*, external carotid artery; *TCD*, transcranial Doppler sonography.

(interquartile range, 1%-4%) on cross-clamping the ipsilateral ECA. On addition of the ICA cross-clamp, there was a further fall in CsO_2 of 3% (ie, a total fall of 6% [3%-9%]). Both these falls were highly statistically significant ($P = .0001$; Fig 1).

The same pattern of highly significant falls occurred in middle cerebral artery blood flow velocity as measured with TCD. The median percentage fall on ECA clamping was 12% (4%-24%), and on ICA clamping the fall was 48% (25%-74%) (Fig 2).

As expected, the fall in regional CsO_2 correlated strongly with the fall in TCD on ICA clamping

(Spearman rank correlation, $r = 0.62$; $P < .01$; Fig 3). The correlation between CsO_2 and TCD on ECA clamping, although less strong, was still statistically significant ($r = 0.32$, $P < .01$; Fig 4). With increasing severity of ipsilateral ICA stenosis, the falls in TCD on external clamping increased from a median of 11.5% (4.1-21.2 interquartile range), if the ICA was less than 90% stenosed, to a median of 13.2% (6-26.9) if the ICA stenosis was 90% or greater, although this difference did not reach statistical significance. There was no correlation between preoperative symptom type and the fall in TCD or CsO_2.

DISCUSSION

These results demonstrate that the ipsilateral ECA contributes at least 10% to 15% of middle cerebral blood flow in patients with severe ICA stenosis. The method used in this study, with sequential cross-clamping first of the ECA and then the addition of an ICA clamp, was likely to underestimate the contribution made by the ECA. As the ECA is clamped, flow in the ICA and in the cross-cerebral collaterals would increase to compensate. It may be possible to accurately measure the proportion of cerebral perfusion supplied by the ECA using a differential injection of a marker into the ECA at the time of surgery. We believed this approach could not be justified and would still be prone to a range of errors.

We could not establish whether the sequence of ECA and ICA clamping affects cerebral perfusion indices differently because it is unethical to clamp the ICA and deliberately delay reperfusion. As the severity of ICA disease increases, we would expect the contribution from the extracranial collateral circulation to be greater. Although our data support this trend in that the falls in middle cerebral artery blood flow velocity and regional CsO_2 on ECA clamping were greater in patients with more severe stenosis of the ICA, the difference was not statistically significant.

There has been much debate on the influence of the extracranial circulation on measurements of near-infrared spectroscopy.[6-9] Clearly, extracranial blood is bound to influence the measurements made by cerebral oximeters that do not include an algorithm to deduct the oxygen saturation in the extracranial circulation. These instruments provide a measure of cerebral oxygenation that may be used to detect global hypoperfusion, but these instruments would not be suitable to study cerebral perfusion during carotid surgery. We have used the Somanetics oximeter, which uses two separate photodetectors placed at differing distances from the light source as a method of calculating CsO_2 in cerebral tissue by deducting the oxygen saturation of blood in the scalp, skull, and superficial brain. Although we would not recommend that this instrument be used as an absolute measure of regional CsO_2 in carotid endarterectomy, the change in percentage saturation is a more valuable indicator of cerebral ischemia than absolute values.

If this technique was substantially influenced by extracranial blood volume, we would not expect to see a simultaneous fall in middle cerebral blood flow measured by TCD on ipsilateral ECA clamping. Not only does middle cerebral blood flow fall on ECA clamping, but this fall in flow was of a similar order of magnitude to the fall in CsO_2. Therefore it must reflect a true fall in cerebral perfusion.

Cerebral emboli may arise through the ECA, and inadequate cross-cerebral collaterals in the context of ipsilateral ICA and ECA stenosis may result in cerebral hypoperfusion. We have performed nine ECA revascularization procedures in symptomatic patients with ICA occlusion not included in this series. All patients had complete resolution of their preoperative symptoms. Our study emphasizes, first, the need to preserve external carotid patency and thereby collateral routes of cerebral perfusion in case of restenosis, and second, the need to consider ECA revascularization in patients with symptomatic ipsilateral ICA occlusion.[10-13]

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